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Role of Intestinal Pathology and Clostridial Species in Clostridial Dermatitis on U.S. Turkey-Grower Farms

Introduction

A disease of turkeys and broilers, clostridial dermatitis (cellulitis/gangrenous dermatitis) has become an issue of concern in recent years. In 2010, the U.S. Animal Health Association (USAHA) ranked clostridial dermatitis among the top three disease issues in turkeys (USAHA, 2010). The disease causes mortality with subcutaneous necrosis, edema, and/or emphysema, and most often affects toms 13 to 18 weeks old (Clark, 2010). Disease pathogenesis, however, is poorly understood. Clostridial dermatitis is believed to be caused by hematogenous transmission of clostridium from the gastrointestinal tract to muscle and skin where bacterial toxins are produced. There is uncertainty about whether pathology of the intestine is required to allow clostridial organisms or their toxins to enter the bloodstream.

Methodology

The U.S. Department of Agriculture's National Animal Health Monitoring System (NAHMS) conducted a study to investigate intestinal pathology and the presence of clostridial organisms on U.S. turkey farms. The goal of the study was to better understand the role of intestinal pathology and clostridial species in the pathogenesis of clostridial dermatitis.

For the study, 15 of the Nation's largest turkey companies were selected to participate. The selected companies represented 76.8 percent of turkeys slaughtered in the United States in 2009 (WATT, 2010). Seven of the selected companies collected biologic samples from 25 farms—19 case farms and 6 control farms.

Participating companies selected case and control farms based on the following criteria:

- Case farm—a farm with a history of frequent clostridial dermatitis outbreaks and on which an outbreak was anticipated during the year;
- Control farm—a farm on which a clostridial dermatitis outbreak was not anticipated.

Case farms were visited weekly during the time leading up to an anticipated outbreak. Control farms were visited weekly during the same time period. Anticipated timing of an outbreak was based on the farm's previous history. Of the 19 case farms, 16 had an outbreak of clostridial dermatitis during the study. Three

case farms and all six control farms did not have an outbreak during the study.

Samples were collected from 397 birds from May 27 through October 16, 2010. Three birds per week were euthanized, and intestinal samples were collected for anaerobic culture and histopathology. Samples of liver, litter, and litter beetles (if present) were also collected weekly for anaerobic culture. Poultry litter samples were taken from three locations in the poultry barns (the middle of the barn, under the feeder, and away from the feeder/drinkers) and then pooled. On the final visit, additional samples collected for culture included spleen and muscle from three euthanized birds.

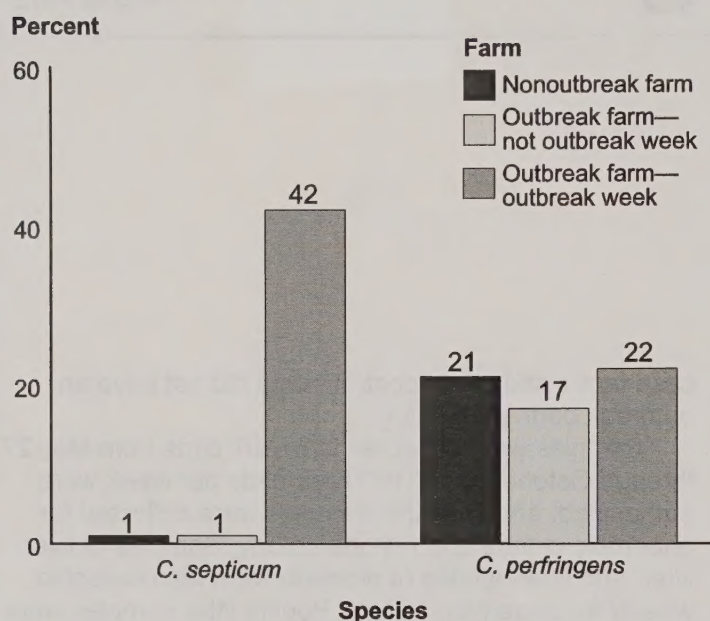
During clostridial dermatitis outbreaks, spleen, liver, and muscle/subcutaneous samples were collected from recent mortalities with lesions, but intestinal culture and histopathology were not performed on birds found dead. See the NAHMS report "Poultry 2010: Clostridial Dermatitis on U.S. Turkey-Grower Farms" for details on the types of samples collected (USDA, 2012). Histopathology was conducted at North Carolina State University and anaerobic culture was conducted at Iowa State University.

The role of *C. septicum* in clostridial dermatitis

Both *Clostridium septicum* and *Clostridium perfringens* have been suggested as causative agents for clostridial dermatitis, but *C. septicum* may be the principal cause (Tellez, 2009).

Findings from this NAHMS study support the theory that *C. septicum* is the primary clostridial organism involved in clostridial dermatitis outbreaks. During outbreaks, 42 percent of sampled birds were positive for *C. septicum* (i.e., had *C. septicum* cultured from one or more tissues). In comparison, only 1 percent of birds from nonoutbreak farms were positive for *C. septicum*. Similarly, in the weeks preceding an outbreak on outbreak farms, only 1 percent of birds were positive for *C. septicum* (figure. 1). These findings suggest that *C. septicum* prevalence increases acutely at the time of an outbreak, as opposed to a high percentage of birds chronically harboring the organism on affected farms.

Figure 1. Percentage of birds positive for *C. septicum* and *C. perfringens*, by farm outbreak status



Approximately 20 percent of birds were positive for *C. perfringens* on outbreak and nonoutbreak farms, regardless of the presence or absence of a clostridial dermatitis outbreak (figure 1). From this study, it is unclear what role, if any, *C. perfringens* plays in clostridial dermatitis.

Positive culture results from liver and spleen samples also provided evidence to support the theory of hematogenous spread of *C. septicum*. In fact, 21 percent of the birds sampled during an outbreak of clostridial dermatitis had *C. septicum* in the liver, and 18 percent had it in the spleen; 48 percent of birds sampled had *C. septicum* in the muscle/subcutaneous tissue (table 1). Of birds euthanized for sampling during an outbreak, 29 percent had *C. septicum* in the muscle/subcutaneous tissues (table 1), but most of these birds did not have gross clostridial dermatitis lesions. Interestingly, no birds had *C. septicum* cultured from the intestines during outbreaks (table 1). However, the presence of *C. septicum* in the liver and spleen still suggests intestines as a likely source of the organism. On outbreak farms in the weeks prior to an outbreak, *C. septicum* was found in only one intestinal sample and in one liver sample (data not shown).

Table 1. Percentage of birds positive for *C. septicum* during a clostridial dermatitis outbreak, by tissue tested and by bird type

Tissue	Percent Birds					
	Euthanized birds		Recent dead with lesions		All birds	
	n	%	n	%	n	%
GI	0/48	0	NA		0/48	0
Liver	2/48	4	15/34	44	17/82	21
Spleen	1/41	2	12/33	36	13/74	18
Muscle	9/31	29	22/33	67	31/64	48
Any	12/48	25	24/37	65	36/85	42

NA= intestine not cultured in birds found dead.

***C. septicum* in poultry litter and litter beetles**

Very few litter or litter beetle samples were positive for *C. septicum* or *C. perfringens*; in fact, only one litter sample from an outbreak farm was positive for *C. septicum* (table 2). This finding was surprising, since litter from flocks with clostridial dermatitis is theorized to contain high numbers of clostridial organisms (Clark, 2010). While litter may contain other species of *Clostridium*, it does not appear that *C. septicum* is present in high numbers in litter, although it might be that *C. septicum* is difficult to recover from litter. This study did not purposefully collect samples from areas where mortalities were removed or where litter was wet, when collecting samples. Litter from these areas might contain higher numbers of clostridial organisms.

Table 2. Number of litter and litter beetle samples positive for *C. septicum* and *C. perfringens*, by farm type

Sample type	Number Samples Positive	
	Farm Type	
	Nonoutbreak	Outbreak
Litter		
<i>C. septicum</i>	0/46	1/71
<i>C. perfringens</i>	0/46	1/71
Litter beetles		
<i>C. septicum</i>	0/22	0/24
<i>C. perfringens</i>	2/22	0/24

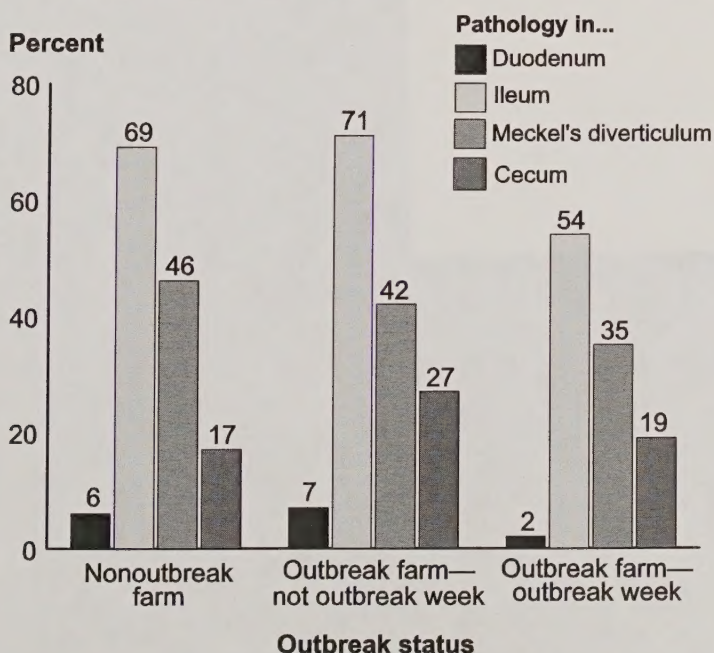
The role of intestinal pathology in clostridial dermatitis

Culture results support the theory of hematogenous spread of clostridial organisms from the intestinal tract; therefore, it is important to understand why or how clostridia get into the bloodstream. Studying the role of intestinal pathology in field outbreaks of clostridial dermatitis is inherently challenging. Ideally, the researcher would collect intestinal samples from birds that are about to break with the disease, to see if intestinal pathology is present before disease onset. However, this is not possible, since intestinal sampling is invasive and the onset of disease in an individual bird cannot be predicted.

This study attempted to measure the role of intestinal pathology by sampling random birds in the weeks leading up to a clostridial dermatitis outbreak. To see if intestinal changes were occurring in the flock, intestinal pathology was assessed microscopically by assigning lesion scores to four areas of the duodenum, four areas of the ileum, and four areas of the cecum. A single lesion score was assigned to Meckel's diverticulum. The presence of luminal bacteria, coccidia, and other parasites was also noted.

Intestinal pathology was common. Over half of all sampled birds had at least mild pathology in the ileum, and about 40 percent of birds had pathology in Meckel's diverticulum. Ileal pathology was most commonly found in the lamina propria or muscularis. A pattern of increasing intestinal pathology in the weeks leading up to an outbreak was not identified. In addition, the percentages of birds with intestinal pathology were similar on outbreak and nonoutbreak farms (fig. 2).

Figure 2. Percentage of birds that had pathology in the following areas of the intestines, by farm outbreak status



Summary

The NAHMS study provided evidence to support two theories about the pathogenesis of clostridial dermatitis:

1. *C. septicum* appears to be important in the pathogenesis of clostridial dermatitis.
2. Hematogenous spread of *C. septicum* occurs on turkey-grower farms during clostridial dermatitis outbreaks.

C. septicum levels increased acutely at the time of an outbreak, as opposed to birds chronically harboring the organism on affected farms. Surprisingly, very few poultry litter or litter beetle samples were culture positive for *C. septicum* or *C. perfringens*.

Over half of turkeys on grower farms had some degree of intestinal damage. Therefore, clostridia may have the opportunity to enter the bloodstream in a high percentage of birds. Although the study results did not clearly define the role of intestinal pathology, intestinal damage may play a role in clostridial dermatitis.

Further research is needed to better understand the complex set of circumstances that culminate in this disease.

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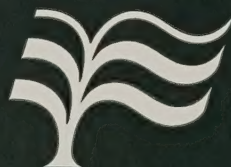


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